Did I Do That? Expectancy Effects of Brain Stimulation on Error-related Negativity and Sense of Agency

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Abstract

■ This study examines the effects of expected transcranial stimulation on the error(-related) negativity (Ne or ERN) and the sense of agency in participants who perform a cognitive control task. Placebo transcranial direct current stimulation was used to elicit expectations of transcranially induced cognitive improvement or impairment. The improvement/impairment manipulation affected both the Ne/ERN and the sense of agency (i.e., whether participants attributed errors to oneself or the brain stimulation device): Expected improvement increased the ERN in response to errors compared with both impairment and control conditions. Expected impairment made participants falsely attribute errors to the transcranial stimulation. This decrease in sense of agency was correlated with a reduced ERN amplitude. These results show that expectations about transcranial stimulation impact users' neural response to self-generated errors and the attribution of responsibility—especially when actions lead to negative outcomes. We discuss our findings in relation to predictive processing theory according to which the effect of prior expectations on the ERN reflects the brain's attempt to generate predictive models of incoming information. By demonstrating that induced expectations about transcranial stimulation can have effects at a neural level, that is, beyond mere demand characteristics, our findings highlight the potential for placebo brain stimulation as a promising tool for research. ■

INTRODUCTION

How would you react if an external device were stimulating your brain through the skull to affect your performance? Would you be more surprised by errors if the device was supposed to improve your performance? What if the device was programmed to hamper your performance? Would you feel less responsible for the errors you committed? In this study, we examine how people respond to errors when they expect that their performance is affected by transcranial brain stimulation.

New technological developments such as neurofeedback, TMS, and transcranial direct current stimulation (tDCS) are attracting attention as tools for cognitive enhancement (O'Connor, Rees, & Joffe, 2012; Sahakian & Morein-Zamir, 2011). Commercial brain devices exploit the widespread belief that humans only use a limited percentage of their brain, which entails the possibility of tapping into unused mental resources (Lilienfeld, Lynn, Ruscio, & Beyerstein, 2011). Indeed, many consumers express a remarkable faith in these techniques to boost their brain capacity (Rusconi & Mitchener-Nissen, 2014) and even highly educated individuals can, for instance, easily be convinced of the mind-reading capacities of a sketchy brain imaging device (Ali, Lifshitz, & Raz, 2014).

Besides emphasizing the importance for brain stimulation studies to take into account double-blind control procedures (i.e., including real vs. sham stimulation), the mere "faith" in brain stimulation techniques also creates interesting possibilities for research on expectancy effects. As such, people's fascination for new brain technologies and the belief in the potential of cognitive enhancement has also been termed "neuroenchantment." It has even been suggested that brain stimulation techniques could act as a "superplacebo," because they optimally exploit people's trust in brain technologies and their motivation to cultivate their own brain potential (Thibault, Lifshitz, & Raz, 2017; Ali et al., 2014).

At the same time, the popular use of brain-based explanations of human behavior increasingly raises ethical issues related to individual responsibility (e.g., "my brain made me do it"; Roskies, 2006; Farah, 2002). These ethical issues become even more prominent with the introduction of neurostimulation techniques that may provide a perfect "excuse" for the externalization of one's actions (Heersmink, 2017; Bostrom & Sandberg, 2009). People might "blame their brain" for their thoughts, experiences, and behavior.

In this study, we elicited expectations of transcranially induced improvement or impairment of performance to assess the effects on prediction error signaling as measured by the error negativity (Ne; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1990, 1991) or error-related negativity (ERN; Scheffers & Coles, 2000; Gehring, Goss, Coles, Meyer, & Donchin, 1993). The Ne/ERN occurs approximately 100 msec after committing an error and has been

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localized to ACC (van Veen & Carter, 2002; Gehring & Fencsik, 2001).[1] ACC activity is assumed to be proportional to the likelihood of committing errors (Alexander & Brown, 2010), and as such, the ERN reflects violated expectations of responding correctly in choice tasks (Holroyd & Coles, 2002), with increased ERN amplitudes for errors that are unexpected (Jessup, Busemeyer, & Brown, 2010; Holroyd, Krigolson, Baker, Lee, & Gibson, 2009; Oliveira, McDonald, & Goodman, 2007; Yasuda, Sato, Miyawaki, Kumano, & Kuboki, 2004; Holroyd, Nieuwenhuis, Yeung, & Cohen, 2003). In this study, we hypothesized that expectations about improved versus impaired cognitive performance through transcranial stimulation should have a directionality-specific effect on the ERN amplitude (i.e., resulting in a stronger vs. reduced ERN amplitude, respectively).

Brain stimulation devices not only affect expectations about performance, they also provide users with the opportunity to attribute unexpected and undesired outcomes to an external cause. We know that cues of external causal factors can moderate the sense of agency over internally generated actions (Chambon, Filevich, & Haggard, 2014; Wegner & Wheatley, 1999) and that such misattributions have been demonstrated in contexts associated with strong expectations, for example, for actions under hypnosis (Polito, Barnier, & Woody, 2013; Woody & McConkey, 2003) and in response to performance enhancing placebos (Clifasefi, Garry, Harper, Sharman, & Sutherland, 2007). Notably, misattributions of agency occur more often in response to negative outcomes, compared with positive or neutral outcomes (Swiney & Sousa, 2013), which may reflect a self-serving bias (Mezulis, Abramson, Hyde, & Hankin, 2004). Negative outcomes are therefore particularly relevant for studying the attribution process in response to expected performance improvement or impairment. In this study, we therefore assessed whether expectations about transcranial stimulation would affect participants' sense of agency indicated by the extent to which unexpected errors would be attributed to the brain stimulation device.

In summary, we manipulated participants' expectations regarding transcranial stimulation using a within-subject design by instructing participants that the brain stimulation would either result in improved, impaired, or unaffected cognitive performance on a cognitive control task (i.e., the Eriksen Flanker task). We hypothesized that expectations about improvement would result in a stronger ERN amplitude in association with errors. Expectations of impaired performance, on the other hand, should result in a reduced ERN amplitude in response to errors. With respect to the sense of agency, we hypothesized that expectations of impaired performance should make participants more likely to externalize errors to the brain stimulation device. Exploratorily, we investigated whether a decreased sense of agency (i.e., attributing errors to the brain stimulation device) was associated with reduced ERN amplitudes to examine whether the strength of prediction error signaling was related to the external attribution of errors. The materials, data, and analysis scripts used for this study are available on the Open Science Framework (see https://osf.io/tzke3).

METHODS

Participants

Initially, 31 healthy participants participated in the experiment (mean age = 32.3 years, range = 18–64 years, 24 women) who received a financial remuneration. Participants were recruited through a local newspaper advertisement (asking people if they would like to participate in a study using new brain stimulation technology to experience the hidden powers of the human mind) as well as through the online participant pool of the University of Amsterdam (for nonpsychology students). Exclusion criteria were a history of brain-related abnormalities and past knowledge of or experience with tDCS. Because of equipment malfunction and/or excessive signal loss, eight participants were excluded from analyses, leaving a final sample of 23 participants (mean age = 28.9 years, range = 18–61 years, 18 women). Specifically, for the first five participants, the tDCS apparatus was not switched off after the sham stimulation. However, it turned out that even though it was not stimulating during the experiment, the apparatus kept leaking current, which heavily distorted the EEG signal. Therefore, these first five participants had to be excluded from analyses. The other three excluded participants were identified upon visual inspection of the EEG signal before the calculation of the ERN. Post hoc power analyses revealed that even after exclusion, the final sample of 23 achieved a power of 90% for the smallest effect of interest (i.e., the ERN effect). The study was approved by the local ethics committee at the Psychology Department of the University of Amsterdam (Project 2016-SP-6649), and all participants were treated in accordance with the Declaration of Helsinki.

Design and Task

A within-subject design was used with the following expectancy conditions: improvement condition, impairment condition, and a neutral (control) condition. An inactive tDCS device (NeuroConn GmbH) was used to increase the credibility of the experimental manipulation. Dependent variables included the ERN amplitude and the sense of agency over committed errors. The order of the different expectancy conditions was counterbalanced across participants, resulting in six different possible sequences of experimental blocks.

The Flanker task (Eriksen & Eriksen, 1974) was used, in which participants were required to report the direction of a centrally presented arrow, to elicit unintended errors, consisting of eight blocks of 20 trials, with 50% congruent trials (central arrow pointing in the same

direction as distractors, i.e., <<<<<) and 50% incongruent trials (central arrow pointing in the opposite direction as distractors, i.e., <<><<), which were repeated over the different expectancy conditions. The task was presented on a 60-cm computer screen (1920 × 1080 pixels) placed at approximately 40 cm from the participant's eyes and was programmed using Presentation software (Neurobehavioral Systems, Inc.). Stimuli were presented in black (font size 36) on a white background. To increase difficulty, the contrast of the central target arrow was reduced (RGB color 235, 235, 235) compared the surrounding distractors.

Before the experimental phase, an individual level of difficulty was determined in a practice phase, to account for large individual differences in accuracy on the Flanker task. This was done by adjusting the response interval (i.e., the window of opportunity of response) to ensure accuracy was within a 60–80% range to obtain a sufficient number of error trials for the calculation of the ERN. The interval was initially fixed at 1000 msec and was shortened with 100 msec after 10 trials if accuracy was higher than 80% or was extended with 100 msec if performance was lower than chance level (50% correct). Thus, the response interval was kept the same if accuracy was within the 60–80% range. If participants failed to respond within the set interval (miss), they received feedback to respond faster. Both errors (incorrect key-press) and misses (no key-press) counted toward the reduction of accuracy. Adjustment continued until performance in two consecutive blocks fell within the 60–80% range, upon which the experimental phase started. During the experimental phase, accuracy was evaluated after each block and adjusted in steps of 50 msec, following the same criteria as during the practice phase. This adjustment was maintained during the experimental phase to eliminate the potential confound that eventual differences in ERN amplitude between conditions could be attributed to the probability of making an error (Holroyd & Coles, 2002). Crucially, by keeping the accuracy stable across conditions, any performance effects would be represented in difference in RTs and thus in the individually set response interval. However, this response interval did not significantly differ between conditions and did thus not confound any condition effects, $F(2, 44) = 0.55$, $p = .580$. In the experimental phase, each error was followed by a feedback screen, in which the participant was required to indicate to what extent the response was influenced by the brain stimulation on a 7-point scale ranging from *not at all* to *completely*, assessing the sense of agency over the error (see trial overview in Figure 1C). This 1–7 scale was recoded in order for high and low values to correspond to high and low sense of agency, respectively.

Expectancy Manipulation

Participants were told that the study aimed to investigate the effect of a completely safe brain stimulation device (tDCS) that has the power to activate the brain's unused potential. The alleged effects of stimulation were strongly emphasized, and the differential effects of the stimulation conditions were explained multiple times. First during a telephone screening and then upon arrival at the lab, the experimental manipulations were verbally disclosed. The researcher verbally repeated the expected effects at the start of each block, and also written information on the induced effects was presented on screen. In the improvement condition, the instruction was as follows: "In this session frontal tDCS is used, which can improve performance. The stimulation releases additional neural activity in your frontal cortex, which makes the neural processing more efficient if you are sufficiently sensitive to the stimulation. The frontal stimulation can make you feel more energized and active." Impairment condition: "In this session parietal tDCS is used, which can impair performance. The stimulation reduces the neural activity in the frontal cortex, which makes the neural processing less efficient if you are sufficiently sensitive to the stimulation. The parietal stimulation can make you feel more tired and dazed." Control condition: "In this session, no stimulation is used." The credibility of the manipulation was enhanced in multiple ways. First, a telephone screening with exclusion criteria based on a standard tDCS screening form was applied, screening participants for a history of epilepsy, severe concussion, psychotropic drugs, pregnancy, and so forth. Second, an actual neuroConn DC stimulator tDCS device and electrodes were used, and sham stimulation (consisting of a 20-sec ramping up of the current as is common practice in real tDCS studies) was administered at the beginning of each stimulation block, so that participants would actually experience a slightly tingling sensation on their head. Last, at the end of the experiment, participants completed a questionnaire on the possible side effects of tDCS.

Procedure

After the study overview and experiment were elaborately described, the participants completed the Flanker task as described above, starting with a practice phase to set the individual level of difficulty. Subsequently, each participant completed the Flanker task in each stimulation condition (i.e., three times) with only the information on the effects of the alleged stimulation differing between conditions. After the two stimulation blocks, participants answered three questions to report their experience during the tDCS, which served as condition-specific manipulation check items ("To what extent do you feel the tDCS affected your performance on the Flanker task?" and "To what extent do you think you have a certain sensitivity for brain stimulation?"—both measured on a 5-point scale from *not at all* to *very much*) as well as to asses subjective performance ("To what extent do you feel your performance on the Flanker became better or worse due to the tDCS?"—measured

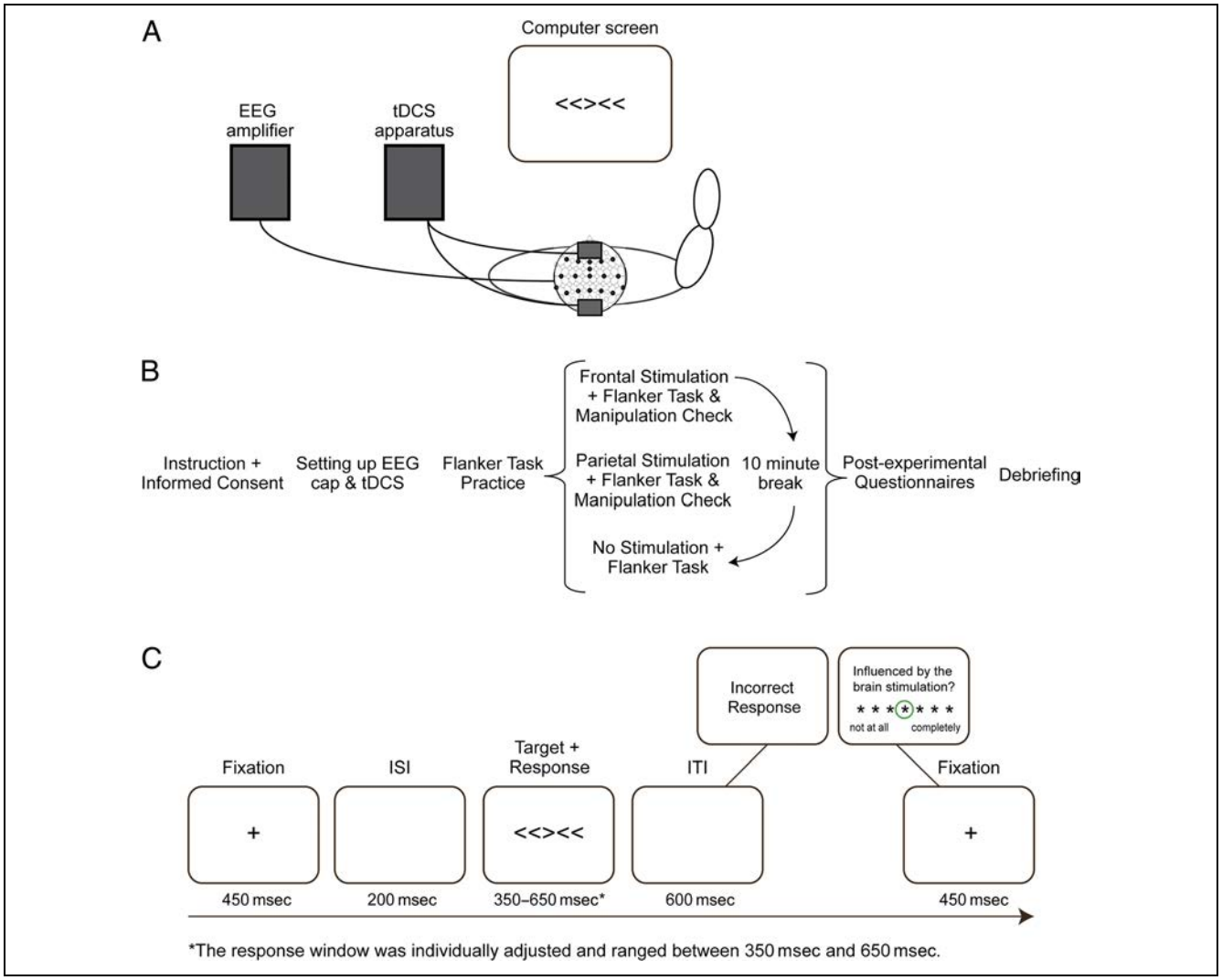

Figure 1. Overview of the (A) experimental setup, (B) procedure, and (C) trial of the used Flanker task. (A) Upon arrival participants were seated at approximately 80 cm from the screen and equipped with the EEG cap and tDCS electrodes. (B) The experimental procedure started with the participants receiving verbal and written instructions about the task and expectancy manipulation and signing an informed consent. EEG and tDCS electrodes were prepared, followed by a practice session of the Flanker task (without stimulation). In the actual test phase, participants completed the Flanker task three times (in randomized counterbalanced order), each time first being informed about the specific stimulation condition (i.e., frontal: improvement; parietal: impairment; no stimulation: no effect). "Active" stimulation sessions were followed by three manipulation check questions on experience of the tDCS. At the end of the experiment, participants completed the postexperimental questionnaires and were debriefed about the placebo manipulation. (C) A trial in the task consisted of a fixation screen (450 msec), a blank screen (ISI; 200 msec), the target presentation, and participants had to respond within the individually determined response interval by pressing the right or left control key with their right or left index finger, respectively. This was followed by an intertrial interval (600 msec). If the given response was incorrect, a feedback screen appeared, and participants had to rate to what extent they believed the error was caused by the brain stimulation device (on a 7-point scale).

on a 5-point scale from *worse* to *better*). This was followed by a 10-min break to "ensure the stimulation had completely worn off" (see Figure 1 for experimental setup, procedure, and task).

After completion of all three conditions, participants filled out the exit questionnaires to assess demographics, their experience during the experiment (as an overall manipulation check; see below), and possible tDCS side effects. In addition, we included a Dutch translation of the Absorption Scale (Tellegen & Atkinson, 1974) with 34 items (Cronbach's $\alpha = .93$) and a translation of the Locus of Control Scale (Rotter, 1966) with 11 items consisting of a pair of statements reflecting an internal versus external locus of control,[2] as both these measures have been related to individual differences in susceptibility to suggestion (Andersen, Schjoedt, Nielbo, & Sørensen, 2014; Groth-Marnat & Pegden, 1998; Paddock et al., 1998).

The overall manipulation check items assessed the influence ("To what extent did you experience the influence of tDCS on the neuronal energy in your brain?") and the efficacy ("To what extent do you consider tDCS an effective method to enhance or impair brain functioning?") of the stimulation. These items were measured on a 5-point scale and ranged from *not at all* to *very much* (see Table 2 for an overview of the condition-specific and overall manipulation check items). In addition, participants

had to describe the purpose of the study in their own words. Finally, participants were carefully debriefed on the true purpose of the study.[3]

Electrophysiological Recordings

EEG was recorded over the entire scalp using 63 electrodes according to the 10/20 system. The data were sampled at 2048 Hz using BioSemi's Active-Two System. Horizontal and vertical EOG were measured with electrodes at the outer canthi and above and below the participant's dominant eye. The frontal tDCS electrode (consisting of a wet 3 × 3 cm sponge electrode) was positioned on top of the EEG cap at the location corresponding to electrode AFz (which was omitted from the EEG), and the parietal tDCS electrode (3 × 3 cm) was positioned in the neck below the EEG cap. Before the start of the experiment, sham tDCS was administered with a neuroConn DC stimulator, including the ramping up for 20 sec with a 1-mA current stimulation with the anodal electrode placed over the pFC and the cathodal electrode in the neck. This sham stimulation was repeated at the beginning of the two stimulation blocks to enhance the credibility of the manipulation. After 20 sec, the device was switched off for the remainder of the block.

The EEG data were down-sampled offline to 256 Hz, re-referenced to the average signal across all electrodes and band-pass filtered between 0.16 and 30 Hz. Response trials (miss trials were excluded from analysis) were segmented into epochs from 100 msec before to 300 msec after the button-press. The mean activity in the window from −100 to 0 msec before response served as baseline and was subtracted from each data point. Data were corrected for ocular movements using the algorithm implemented in Brain Vision Analyzer (Gratton, Coles, & Donchin, 1983). A semiautomatic procedure was used to detect and reject artifacts. The criteria applied were a voltage step of more than 50 μV between sample points, a voltage difference of 200 μV within a trial, amplitudes where the signal exceeded the −100 and +100 μV threshold, and a maximum voltage difference of less than 0.5 μV within a 100-msec interval. Intervals were flagged and manually removed. For the final sample of 23 participants, this processing resulted in an average of 1.3% (*SD* = 1.2%) of the response trials being rejected from analysis due to artifacts. Individual ERPs were calculated separately for correct and incorrect trials and for each stimulation condition (improvement, impairment, control), resulting in six ERPs per participant.

For each incorrect trial, the ERN was quantified as the activity at the peak of the signal using automatic peak selection for a negative deflation within the predefined time window ranging from 0 to 150 msec at electrodes FCz and Cz. In addition, for correct trials, the correct response negativity (CRN) was extracted in the same way, using the same time window and electrode sites. As visual inspection revealed the strongest ERN at location FCz (see Figure 2E), in line with existing literature (Falkenstein,

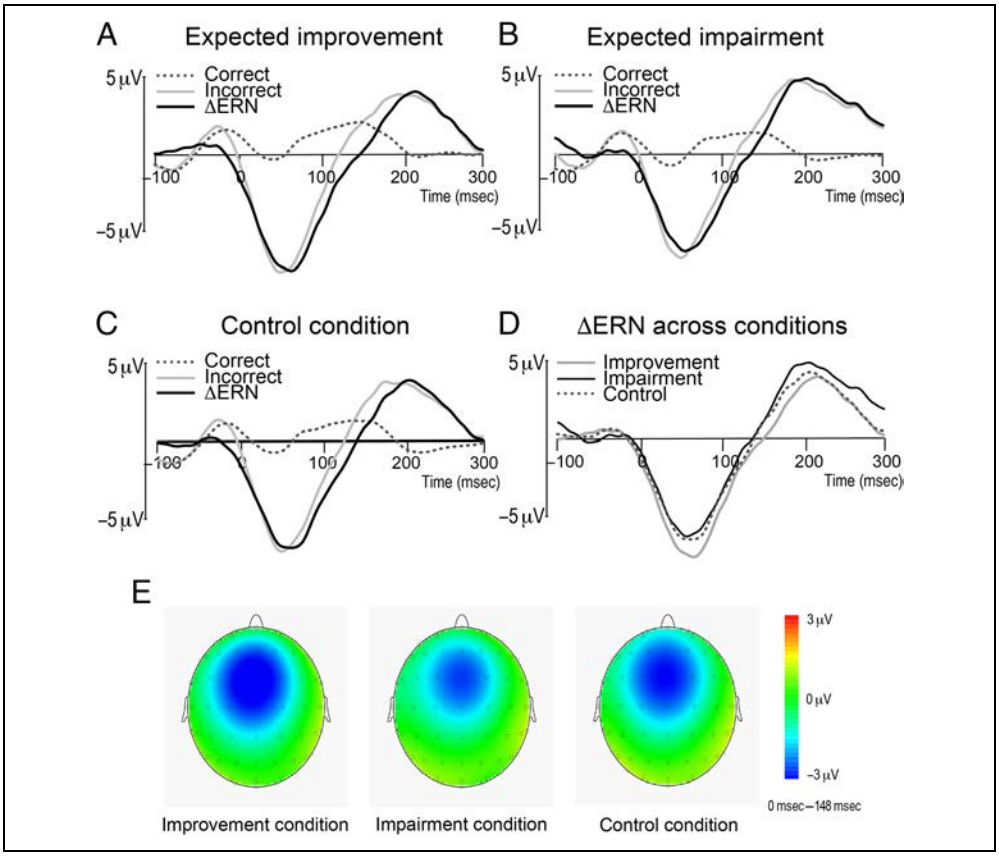

Figure 2. Response-locked ERPs at FCz following correct (dotted lines) and incorrect responses (gray lines) on the Flanker task and the difference between both responses (ΔERN; black lines) for participants in the (A) improvement, (B) impairment, and (C) control conditions, showing a clear ERN response across all conditions. (D) Comparison of ΔERN waveforms in the improvement, impairment, and control condition showed a larger ΔERN amplitude for the improvement compared with the impairment and control condition. (E) Voltage topographical maps of the ΔERN-related activity within the 0–150 msec time window per condition, showing the strongest activity at the location of electrode FCz.

Hoormann, Christ, & Hohnsbein, 2000; Gehring et al., 1993), the ERN peak amplitude at this electrode was used in further analyses. We note, however, that selecting electrode Cz yielded similar results as reported in the main analysis in this manuscript.

Statistical Analyses

For the ERP data, a repeated-measures ANOVA with Condition (i.e., improvement vs. impairment vs. control) and Correctness (i.e., correct vs. error trials) as within-subject factors was performed to validate a main effect of Correctness and hence a substantial ΔERN. This ΔERN (i.e., the difference between the ERN and the CRN, which was obtained by subtracting the mean CRN from the mean ERN) is taken to reflect the neural response to errors specifically and remove the effect of generic response monitoring that is manifested in both ERN and CRN (Simons, 2010). The remaining analyses in this manuscript therefore focused on the ΔERN.

The ΔERN was compared between conditions using Bonferroni-corrected paired t tests. Similarly, a repeated-measures ANOVA with Condition (improvement vs. impairment vs. control) as within-subject factor was performed for the sense of agency, followed by Bonferroni-corrected paired t tests to investigate post hoc contrasts. In addition, the correlation between the sense of agency and ΔERN data was calculated to examine whether attribution of errors was related to the strength of the prediction error signal (i.e., the ΔERN).

The subjectively perceived efficacy of the experimental expectancy manipulation was checked by comparing (1) the condition-specific manipulation check items between both stimulation blocks and (2) the subjective performance evaluation between both blocks by using a one-tailed t test (as we had a priori predictions regarding the directionality of the effects). In addition, to eliminate the possibility that differences in objective performance (i.e., differences in accuracy or speed of responding) could eventually underlie the effects that we observed, two repeated-measures ANOVAs with Congruency (congruent vs. incongruent trials) and Condition (improvement vs. impairment vs. control) as within-subject factors were conducted with the percentage of errors and RTs as dependent measures.

RESULTS

Confirmatory Results

Error-related Negativity

The ERPs for the different experimental conditions are represented in Figures 2 and 3A. To study the effects of our expectancy manipulation on the ERN in response to error trials on the Flanker task, we first conducted a repeated-measures ANOVA with Condition and Correctness as within-subject factors. As expected, a main effect of Correctness was found, with the ERN amplitude being significantly larger than the CRN amplitude, $F(1, 22) = 100.77$, $p < .001$, $\eta_p^2 = .821$. The main effect of Condition on the ERP amplitude was not significant, $F(2, 44) = 2.96$, $p = .062$. Importantly, we found a significant interaction effect between Condition and Correctness, $F(2, 44) = 6.79$, $p = .003$, $\eta_p^2 = .236$, suggesting that the ΔERN differed between the different expectancy manipulations. Separate analyses for correct and error trials (i.e., the CRN and ERN, respectively) indicated that the interaction effect was driven by error trials, as an ANOVA for the ERN indeed showed a significant effect for Condition, $F(2, 44) = 5.05$, $p = .011$, $\eta_p^2 = .187$, whereas the ANOVA for the CRN did not, $F(2, 44) = 1.93$, $p = .157$. Notably, in addition to being nonsignificant, the direction of amplitudes for the conditions was different for the CRN, namely impairment < improvement < control, rather than improvement < control < impairment as found for the ERN. To further explore this interaction effect, the ΔERN was directly compared between conditions by means of three post hoc Bonferroni-corrected paired t tests. We found a significant difference between the improvement ($M = -7.90$ μV) and impairment ($M = -6.03$ μV) condition in ΔERN amplitude, $t(22) = -3.64$, $p = .004$, Cohen's $d = 0.76$, as well as between the improvement and the control condition ($M = -6.69$ μV), $t(22) = -2.66$, $p = .043$, Cohen's $d = 0.55$. No difference was found between the impairment and the control condition in the ERN amplitude, $t(22) = 1.17$, $p = .77$. These findings indicate that participants showed a stronger neural response to violations of enhanced performance expectations, compared with impaired performance expectations or unaffected performance expectations. Following up on the most relevant analysis—the ΔERN between conditions, we computed Bayes factors to quantify the evidence for the expectancy effect on the ΔERN using the statistics software JASP (JASP Team, 2018). That is, we used a Bayesian repeated-measures ANOVA with default Jeffreys–Zellner–Siow priors for ANOVA, using r-scale of fixed effects (condition) of 0.5 and r-scale of random effects (subject; within factor) of 1 (Rouder, Morey, Speckman, & Province, 2012; Wetzels, Grasman, & Wagenmakers, 2012). The results indicated strong evidence for the effect of expectancy condition on the ΔERN; $BF_{10} = 13.52$, with post hoc test revealing $BF_{10} = 25.65$ for improvement versus impairment, $BF_{10} = 3.63$ for improvement versus control, and $BF_{10} = 0.40$ for impairment versus control, reflecting strong evidence for difference, moderate evidence for difference, and anecdotal evidence against difference in ΔERN, respectively[4] (Lee & Wagenmakers, 2014).

Sense of Agency

A significant main effect of Condition on sense of agency was observed, $F(2, 44) = 59.94$, $p < .001$, $\eta_p^2 = .731$ ($BF_{10} = 2.42 \times 10^{12}$; extreme evidence for the expectancy condition hypothesis), with post hoc tests revealing that all expectancy conditions significantly differed from each other (see Figure 3B); the sense of agency was significantly higher in the control condition ($M = 6.82$, $SD = 0.35$)[5] than

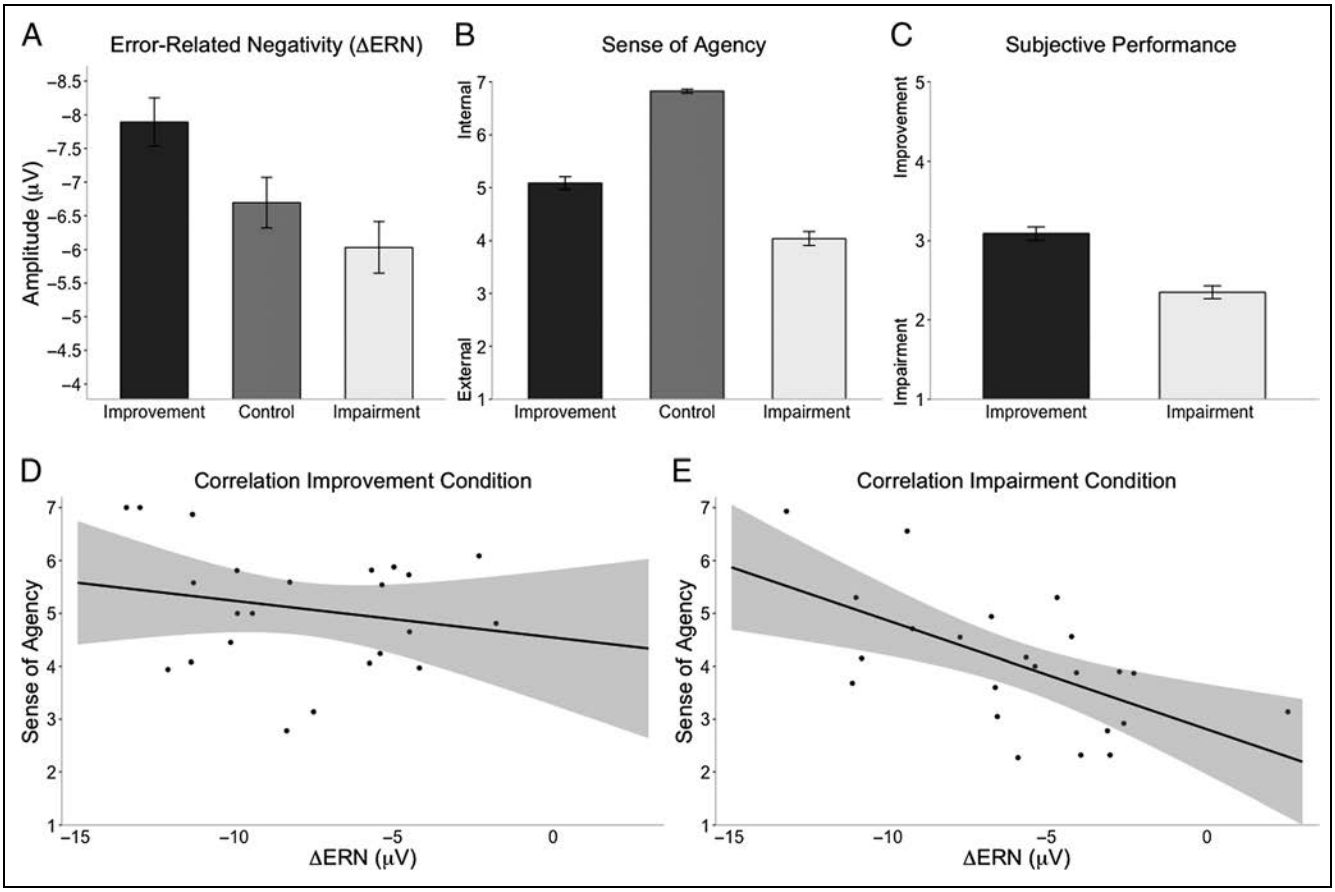

Figure 3. Graphs depicting the main results per condition. (A) ERN (ΔERN) amplitudes, reflecting the strongest prediction error response in the improvement compared with the control and impairment condition. (B) Sense of agency over committed errors, showing a lowered sense of agency in the experimental conditions, and especially more external attribution (i.e., lower sense of agency) in the impairment condition, compared with the improvement condition. (C) Subjective performance effects of the transcranial stimulation, demonstrating the difference in subjectively experienced performance as a function of the expectancy manipulation. Scatterplots of the ΔERN and the sense of agency in the improvement (D) and impairment (E) condition, displaying the absence of a correlation in the former and a significant negative correlation in the latter. Specifically, in the impairment condition, a stronger ΔERN amplitude was associated with more internal attributions (i.e., higher sense of agency) and a smaller ΔERN amplitude with more external attributions (i.e., lower sense of agency). In this figure, the ΔERN is represented as the ERN difference wave (i.e., incorrect – correct trials). Error bars indicate standard errors.

in the improvement condition ($M = 5.09$, $SD = 1.16$), $t(22) = 6.99$, $p < .001$, Cohen's $d = 1.46$ ($BF_{10} = 32565$; extreme evidence for the difference hypothesis) and than in the impairment condition ($M = 4.04$, $SD = 1.24$), $t(22) = 9.87$, $p < .001$, Cohen's $d = 2.06$ ($BF_{10} = 7.41 \times 10^6$; extreme evidence for the difference hypothesis; see Table 1 for descriptives). Crucially, the sense of agency in the impairment condition was significantly lower than in the improvement condition, $t(22) = -4.41$, $p < .001$, Cohen's $d = 0.92$ ($BF_{10} = 134.4$; extreme evidence for the difference hypothesis).

Manipulation Checks

Our data indicated that the placebo tDCS manipulation was successful, as the mean scores on the overall manipulation check items were $M = 3.26$ ($SD = 1.01$) for tDCS influence and $M = 3.13$ ($SD = 0.76$) for tDCS efficacy, indicating that

Table 1. Descriptive Statistics for Subjective Experience of Performance, Sense of Agency, and Objective Flanker Performance

	Improvement	*Impairment*	*Control*
Subjective performance[a]	3.09 (0.79)	2.35 (0.76)	
Sense of agency[b]	5.09 (1.16)	4.04 (1.24)	6.82 (0.35)
Objective performance			
Errors (%)			
Congruent	1.6 (1.74)	1.5 (1.81)	1.4 (1.97)
Incongruent	25.5 (9.36)	25.5 (7.34)	27.9 (8.60)
RTs			
Congruent	337 (37.6)	339 (35.6)	339 (36.0)
Incongruent	396 (43.1)	399 (43.1)	397 (46.4)

Displays mean values, with standard deviations given in parentheses.

[a]Measured on a 5-point scale.

[b]Measured on a 7-point scale.

Table 2. Overview of the Manipulation Check Items per Condition and Overall

		Improvement	*Impairment*
Condition-specific Manipulation Check Items			
Influence	*To what extent do you feel the tDCS affected your performance on the Flanker task?*	3.00 (0.66)	3.32 (0.87)
Sensitivity	*To what extent do you think you have a certain sensitivity for brain stimulation?*	3.00 (0.79)	3.17 (0.83)
Subjective performance	*To what extent do you feel your performance on the Flanker became better or worse due to the tDCS?*	3.09 (0.79)	2.35 (0.76)
Overall Manipulation Check Items			
Influence	*To what extent did you experience the influence of tDCS on the neuronal energy in your brain?*	3.26 (1.01)	
Efficacy	*To what extent do you consider tDCS an effective method to enhance or impair brain functioning?*	3.13 (0.76)	

Displays mean values, with standard deviations given in parentheses. Measured on a 5-point scale.

participants judged the brain stimulation to have exerted "moderate" to "substantial" influence on their performance and be moderately to substantially effective as a method to enhance or impair brain functioning.

Importantly, the extent of the experienced influence and sensitivity did not differ between the improvement ($M = 3.00$, $SD = 0.66$; $M = 3.00$, $SD = 0.79$) and impairment ($M = 3.32$, $SD = 0.87$; $M = 3.17$, $SD = 0.83$) condition, as indicated by comparison between the condition-specific manipulation check items after the stimulation blocks on influence, $t(22) = -1.43$, $p = .167$ and on sensitivity, $t(22) = -0.85$, $p = .406$, indicating that participants felt equally influenced by and sensitive to the tDCS in both the improvement and impairment condition (see Table 2).

As expected, the items assessing subjective experience of improvement/impairment in performance showed a significant difference between experimental conditions, $t(22) = 3.36$, $p = .001$, Cohen's $d = 0.70$, indicating that, in the improvement condition, participants rated their subjective performance as better ($M = 3.09$, $SD = 0.79$) compared with the impairment condition ($M = 2.35$, $SD = 0.76$), on a scale ranging from 1 = *impairment* to 5 = *improvement* (see Table 1 and Figure 3C).

Exploratory/Additional Results

Sense of Agency and ΔERN

In the impairment condition, committing errors and attributing these to the brain device is congruent with participants' expected effects of the brain stimulation (as opposed to the improvement condition in which committing errors contradicts the expected effect of brain stimulation). A significant negative correlation between the sense of agency and the ΔERN amplitude was found in the impairment condition, $r(21) = -.60$, $p = .002$ (two-tailed), indicating that an internal sense of agency was related to a larger ΔERN amplitude and an external sense of agency to a smaller ΔERN amplitude (see Figure 3E). No such correlation was found in the improvement condition, $r(21) = -.21$, $p = .35$ (two-tailed; see Figure 3D), or in the control condition, $r(21) = .16$, $p = .46$ (two-tailed). Although in the same direction, the correlation coefficients for the impairment and improvement condition were found to differ significantly, using Fisher's *Z*-transformation for comparison of nonoverlapping correlations based on dependent groups, $z = 1.97$, $p = .049$ (Raghunathan, Rosenthal, & Rubin, 1996). Moreover, analysis using Bayes factors further corroborates the respective presence and absence of a correlation between ΔERN and sense of agency in the impairment and improvement condition; for the impairment condition $BF_{10} = 20.55$, indicating strong evidence in favor of the correlation hypothesis, whereas in the improvement condition $BF_{10} = 0.394$, which reflects anecdotal evidence for the null hypothesis assuming no relation between ΔERN and sense of agency.

In addition, we exploratorily assessed the correlations between the ΔERN, sense of agency, subjective performance, and condition-specific manipulation check items on experienced influence and subjective sensitivity for tDCS. As can be seen in Table 3, most items were correlated in the impairment condition, indicating that, with regard to suggestions of impairment, participants who felt most influenced by the stimulation also had a larger ΔERN amplitude, lower sense of agency over errors, judged their performance to be worse and indicated to be more sensitive to the effects of tDCS, relative to participants that felt less influenced by the tDCS. In the improvement condition, none of these correlations appeared.

Table 3. Pearson Correlations for ERN Amplitude, Sense of Agency, Subjective Experience of Performance Modulation, Experienced tDCS Influence, and Subjective Sensitivity for tDCS Displayed for the Improvement (above Diagonal) and Impairment (below Diagonal) Conditions

	ΔERN	*Sense of Agency*	*Subjective Performance*	*Experienced Influence*	*Subjective Sensitivity*
ΔERN	—	−.206	.064	−.063	−.088
Sense of agency	−.604**	—	.232	−.230	−.129
Subjective performance	−.145	.432*	—	.000	−.072
Experienced influence	.430*	−.618**	−.587**	—	.189
Subjective sensitivity	.159	−.258	−.590**	.659***	—

Intercorrelations for the improvement condition are presented above the diagonal, and intercorrelations for the impairment condition are presented below the diagonal.

*$p < .05$.

**$p < .01$.

***$p < .001$.

Temporal ΔERN Effects

To assess the temporal development of the ΔERN over the course of the experiment, we calculated the ΔERN per two blocks (i.e., four sections) for each condition and each participant.[6] The results revealed no main effect for Section of the experiment, $F(2.03, 44.47) = 1.46, p = .244$ (Greenhouse–Geisser corrected for violation of the assumption of sphericity), nor an interaction between Section and Condition, $F(6, 132) = 0.36, p = .903$. Figure 4 illustrates the absence of this main effect and interaction.

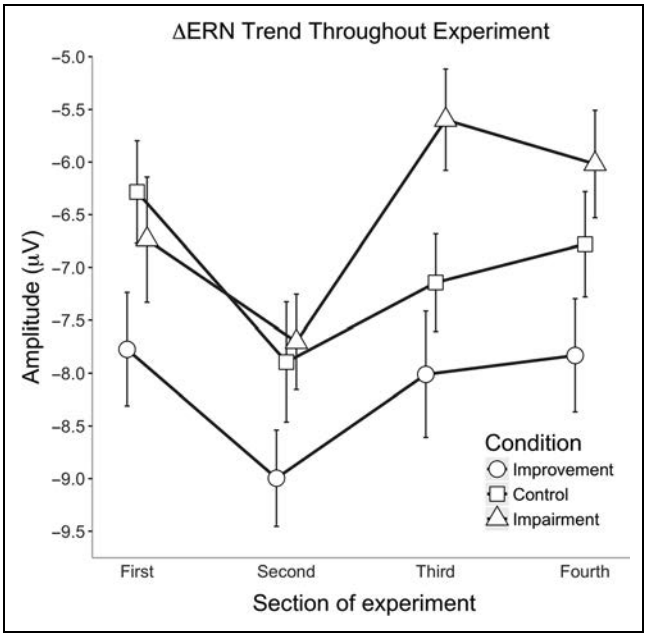

Figure 4. Graphical representation of the temporal development of the ΔERN throughout the experiment per condition, showing the absence of an interaction between section and condition, that is, the ΔERN amplitude remains higher in the improvement condition relative to the control and impairment condition.

Objective Performance Effects

The absence of objective performance differences between conditions was confirmed by additional analysis of the behavioral data (see Table 1 for descriptives). With regard to errors, there was no main effect for Condition, $F(2, 44) = 1.79, p = .178$, indicating that participants performed equally well in the improvement, impairment, and control condition—logically, as the task difficulty was kept constant. The main effect for Flanker congruency was significant, $F(1, 22) = 241.12, p < .001, \eta_p^2 = .916$, indicating that people made more errors on incongruent (26.3%) than on congruent trials (1.5%), but there was no significant interaction effect between Condition and Congruency, $F(2, 44) = 2.55, p = .09$ (BF_{01} interaction = 8.26; moderate evidence for the null hypothesis). Moreover, the ratio between errors (average: $M = 13.9\%$, $SD = 4.0\%$) and misses (average: $M = 15.0\%$, $SD = 3.7\%$) did not significantly differ between conditions, $F(1.50, 33.03) = 1.63, p = .215$ (the Greenhouse–Geisser correction was used, as Mauchly's test indicated that the assumption of sphericity was violated). Similarly, for the RTs, the repeated-measures ANOVA revealed no main effect for Condition, $F(2, 44) = 0.30, p = .739$, and a strong effect for Congruency, $F(1, 22) = 360.23, p < .001, \eta_p^2 = .942$, showing that people were significantly slower on incongruent (397 msec) compared with congruent (339 msec) trials. There was again no interaction effect between Condition and Congruency, $F(2, 44) = 0.24, p = .786$ (BF_{01} interaction = 23.81; strong evidence for the null hypothesis). These behavioral results effectively rule out the potential confound that performance differences (i.e., RTs or accuracy) could have contributed to our effects.

Order Effects

When the between-subject factor of Order of expectancy conditions was added to the ANOVAs for ΔERN or the

sense of agency, no significant effects were found. Specifically, for ΔERN amplitude, the main effect of Order was nonsignificant, $F(5, 17) = 1.50$, $p = .242$, as was the interaction effect between ΔERN and Order, $F(10, 34) = 1.67$, $p = .346$. For the sense of agency, there was marginally significant main effect of Order, $F(5, 17) = 2.71$, $p = .056$. Crucially, the interaction between Condition and Order was nonsignificant, $F(10, 34) = 1.21$, $p = .376$.

Individual Difference Measures

Individual differences in level of absorption and locus of control as measured by the absorption scale and the locus of control scale did not correlate with any of the main dependent measures (i.e., ΔERN, sense of agency) nor with the subjective experience of performance and objective performance. Further analyses on individual differences were therefore not conducted.

DISCUSSION

In this study, we manipulated expectations of transcranial stimulation to investigate expectancy effects on the ERN and the sense of agency by suggesting improved, impaired, or no cognitive performance modulation. In doing so, our study was the first to relate placebo brain stimulation to neural prediction error signaling.

In both the impairment and the improvement condition, participants were more likely to falsely attribute errors to the brain stimulation device. As expected, the misattribution of agency was strongest when the device was supposed to hamper performance, corroborating previous evidence that misattributions of agency are especially prominent when potential external sources can intuitively be used as an "excuse" for failures (Moore, Wegner, & Haggard, 2009; Aarts, Custers, & Wegner, 2005). That is, the stimulation device may give users the experience of being less responsible for unexpected negative outcomes, paralleling findings for actions performed under hypnosis (Polito et al., 2013; Woody & McConkey, 2003) and expected thought insertion (Swiney & Sousa, 2013). Admittedly, in the improvement condition, the rating of one's sense of agency over errors was more ambiguous, as committing errors countered the expected positive effect of the stimulation. This ambiguity in the improvement condition poses a limitation on this study, and we suggest that future studies might additionally assess agency over successes rather than just failures to more consistently investigate feelings of responsibility for successful performance under placebo brain stimulation.

Our findings in the impairment condition, nevertheless, suggest that the externalization of responsibility is not merely a post hoc attribution. That is, when external attribution of errors was congruent with the raised suggestions (i.e., expected impairment), the tendency to misattribute errors to the brain stimulation was found to be associated with a decreased ERN amplitude. This suggests that prior expectations about external influences affect error processing already at a very early stage, and thereby, this finding corroborates previous evidence that the ERN amplitude is affected by the perceived responsibility for and concern about one's actions, as has for instance been shown for free will manipulations (Rigoni, Wilquin, Brass, & Burle, 2013) and religious priming (Good, Inzlicht, & Larson, 2015). Interestingly though, we did not find that the ERN was lower at the group level in the impairment condition compared with the control condition. Rather, the degree to which behavioral errors elicited prediction errors appeared to depend on the extent to which individuals externalized agency to the stimulation device.

In the improvement condition, on the other hand, there seemed to be a general expectancy effect. That is, when expecting fewer errors, violation of expectations (i.e., still committing errors) elicited stronger neural prediction error signals. It has been suggested that the ERN reflects a violation of expected positive outcomes (Wessel, Danielmeier, Morton, & Ullsperger, 2012; Holroyd et al., 2003, 2009; Compton et al., 2007; Holroyd, Larsen, & Cohen, 2004; Yasuda et al., 2004), and accordingly our findings indicate a stronger prediction error signal when expectations about enhanced cognitive performance are violated. More broadly, our findings corroborate the error likelihood hypothesis of ACC, which posits that ACC activity is proportional to the likelihood of errors (Alexander & Brown, 2010; Jessup et al., 2010).

We argue that these findings can be accounted for by the predictive processing model (Buchel, Geuter, Sprenger, & Eippert, 2014; Clark, 2013; Kilner, Friston, & Frith, 2007), according to which placebo effects can be elicited when prior expectations are high and when sensory data (moderation of accumulative performance in this case) are low in precision. Note that these effects were not confounded by actual performance differences, as the error ratio and RTs were equal across all expectancy conditions. The effects demonstrated by our manipulation checks of subjective experience indicate that, despite the absence of objective performance differences between conditions, participants still sustained their belief in the efficacy of the device. Moreover, the ERN amplitude did not decrease over the course of the experiment and remained higher upon suggested improvement compared with suggested impairment throughout the entire block. This indicates that the errors experienced in the enhancement condition did not result in participants revising their prior beliefs and expectations. Although people may use outcome probability as determined by actual error frequency to update their expectations (Nassar, Wilson, Heasly, & Gold, 2010), a priori beliefs may still overrule the effects of actual outcome probability and hence affect prediction error signals (Sharot, Riccardi, Raio, & Phelps, 2007). Exactly why participants did not adjust their expectations of error likelihood remains unclear. Perhaps the instructions stating that the strength of the stimulation might fluctuate over

the course of the experiment enhanced participants' persistent expectations. In addition, the speed of the task may have prevented them from reflecting on perceived effects and from updating their beliefs accordingly until afterwards (i.e., at the end of the experimental task).

Furthermore, the ERN has also been interpreted as a neural distress or worry signal, being sensitive to the emotional value of errors (Maier & Steinhauser, 2016; Moser, Moran, Schroder, Donnellan, & Yeung, 2013; Inzlicht & Al-Khindi, 2012; Inzlicht & Tullett, 2010; Hajcak, Moser, Yeung, & Simons, 2005; Gehring & Willoughby, 2002). As such, the stronger ERN in the improvement condition could also reflect that participants were more upset or frustrated by errors when expecting enhanced cognitive performance, which perhaps motivated them to put in even more effort to experience the power of the brain stimulation, rather than giving up and adjusting their expectations about the brain stimulation. We are well aware of the fact that the interpretation about the precise nature of the ERN and its functional significance remain a matter of ongoing debate—and we suggest that future studies could be designed to disentangle the violation of expectation from the distress account (e.g., by independently manipulating expectations about the likelihood and the reward value of errors).

Either way, the sustained effects of our placebo manipulation are remarkable, especially because participants did not experience actual performance improvement when assessing their performance afterwards. The open questions related to the experience during the improvement condition indicated that 16 of the participants (70%) felt more focused/concentrated/alert/awake or faster in this condition, whereas seven participants (30%) felt nothing or only frustration. Interestingly, only five participants reported solely positive effects. Eleven of the 16 participants who reported some experiences in line with the suggestion also provided a counterargument as to why their actual performance might not have been better, for example, "I was better able to concentrate, but therefore I focused more on the correct answer and therefore it took longer" or "I felt like I registered faster in which direction the arrow was pointed, but my fingers didn't necessarily follow." We cautiously interpret this as suggesting that people can quite easily be convinced of some induced placebo effect, yet if the to-be-affected outcome is too salient, as was perhaps the case for errors on the Flanker task, they might themselves come up with alternative explanations justifying the observed results—at least when reflecting on their performance afterwards.

In addition, the absence of a relationship between the ERN and the sense of agency in the improvement condition may reflect a dissociation between implicit and explicit processing of agency (Moore, Middleton, Haggard, & Fletcher, 2012; Pacherie, 2007). The ERN may be considered an automatic response to errors (i.e., occurring rapidly and outside volitional control), whereas the explicit judgment of agency may result from a postdictive process in which errors can be attributed to an internal or an external source. Following Wegner's account of reconstructive agency, this postdictive attribution can be understood as a process in which people seek explanations for their perceived actions and thoughts after they occurred (Synofzik, Vosgerau, & Voss, 2013; Wegner, 2003). These implicit and explicit agency processes often coincide—stronger ERN with higher agency rating, as in the impairment condition, but they can also diverge—as in the improvement condition. In the latter case, the ERN amplitude and the sense of agency rating may respectively reflect the effects of implicit expectations that one should perform better due to the brain stimulation and the explicit belief that the stimulation was also responsible for any committed errors.

Across the board, it seemed that people did in fact experience some effect of the stimulation but also realized that they still made several errors and therefore came up with explanations to reconcile both experiences. To reduce the salience of this discrepancy, we suggest that future placebo studies should specifically focus on subjective effects in the cognitive domain, for example, by considering a task that encompasses more opaque outcomes, for instance, focusing on speed, or using a motoric task, in which outcomes are arguably more subjective. In this study, however, our main focus was on the ERN, rather than subjective performance effects.

Finally, to maximize expectancy effects, we exploited multiple suggestive cues including a real tDCS device, verbal suggestions, a lab setting, and screening materials combined with actual recording of brain data (cf. Andersen et al., 2014). Indeed, verbal suggestions, physical context and reliability, status, or authority of the experimenter have all been shown to contribute to enhance placebo responses (Howe, Goyer, & Crum, 2017; Schjoedt, Stødkilde-Jørgensen, Geertz, Lund, & Roepstorff, 2011; Di Blasi, Harkness, Ernst, Georgiou, & Kleijnen, 2001; Crow et al., 1999). Moreover, when commercial brain stimulation devices are used in a naturalistic setting, verbal information and the expertise and trustworthiness of the provider are also mainly employed to boost efficacy expectancy and optimize outcomes.[7] That being said, an interesting next step would be to disentangle the relative contribution of each of the specific components, for instance, by comparing the present paradigm to other brain-related placebo manipulations such as neurofeedback (e.g., Thibault et al., 2017) or "frequency stimulation" (e.g., Schwarz & Buchel, 2015) and to traditional placebo manipulations such as pills or creams. Interestingly, in our study we found that mere verbal suggestions were already sufficient to reliably induce placebo effects—in contrast to previous studies that have used conditioning or reinforcement learning in combination with verbal suggestions to induce placebo responses (e.g., Schwarz & Buchel, 2015).

In conclusion, this study demonstrates that sole expectations about transcranial stimulation may have profound effects on neural error processing and on the attribution

of errors to an external source. These findings emphasize the need to be aware of the ethical consequences when people are able to "blame the brain" for their performance or experiences, especially outside the lab. More importantly, this study also demonstrates the other side of the coin, namely, the potential for brain stimulation paradigms to be used in cognitive (placebo) research, aligning with previous applications of placebo brain stimulation to induce analgesia (Krummenacher, Candia, Folkers, Schedlowski, & Schonbachler, 2010) and even mystical experiences (van Elk, 2015; Andersen et al., 2014; Granqvist et al., 2005). Indeed, the fact that induced expectations about transcranial stimulation have effects at a neural level, that is, beyond mere demand characteristics and subjective effects, advocates placebo brain stimulation or neuroenchantment manipulations as a promising tool for novel experimental manipulations.

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Notes

1. Although there has been discussion on the precise nomenclature of this ERP component (see, for instance, Gehring et al., 1993; Falkenstein et al., 1990), there is agreement that Ne and ERN denote the same component (Falkenstein et al., 2000). Throughout this article, we will refer to the negative peak component following an error as the ERN.
2. Analysis indicated that the reliability of the Locus of Control Scale was relatively low (Cronbach's $\alpha = .52$). Deletion of the three items with the lowest loading on the first factor in a factor analysis (PCA) resulted in a moderately reliable Locus of Control Scale ($\alpha = .65$) with eight items.
3. Specifically, they were explained that "your brain was not actually stimulated and that all experiences you had were self-generated. Placebo has been proven to be a powerful effect for instance in medical practice and we are now looking at the effects of placebo brain stimulation on cognitive performance. We also looked at the influence on brain processes to assess the underlying mechanisms of the placebo effect. The information you received at the beginning of the experiment is factually accurate. tDCS is indeed a method to activate and deactivate the brain and influence performance. We did however not do this in the current experiment."
4. Note that the Bayes factor for the post hoc tests is uncorrected for multiple comparisons.
5. The fact that the sense of agency rating was not exactly 7.0 might seem strange, as there was no external influence to reduce the sense of agency. Indeed, only 10 participants had a mean agency rating in the control condition of exactly 7.0. Of the other 13, 11 pressed 6 a few times, either by accident or on purpose, perhaps because they still felt the wires were "doing something." Importantly, only two participants had an average below 6.0 in the control condition, which can be attributed to the fact that the default indicator started at 4, and these people realized only about halfway through the block that they were to shift the indicator for "no influence of the stimulation." Excluding these two participants did not change any results of the main analyses (i.e., ΔERN, sense of agency).
6. We note that, in order to extract the information for the four sections, we had to reanalyze the data in Brain Vision Analyzer. Because of a different distribution of trials over conditions, the results of this exploratory analysis may therefore differ slightly from the main analyses, although the conclusions remain the same (i.e., a significant effect for condition, $F(2, 44) = 6.21$, $p = .004$, $\eta_p^2 = .220$).
7. See for instance the quote by Dr. Williams on the Web site of the commercial tDCS provider *foc.us* (https://www.foc.us/). Notably, this device was in fact shown to impair rather than improve working memory in a placebo-controlled study (Steenbergen et al., 2016).

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